

ABSTRACT

Regulation of expression of CTL activity by macrophage migration inhibitory factor (MIF) is disclosed. In a mouse model using the EL4 tumor, cultured splenocytes from tumor-primed mice secrete high levels of MIF following antigen stimulation *in vitro*. Parallel splenocytes treated with neutralizing anti-MIF mAb showed a significant increase in CTL response against tumor cells compared to control mAb-treated cultures, with elevated expression of IFN γ . Histology of tumors from anti-MIF treated animals showed increases in infiltration of both CD4⁺ and CD8⁺ T cells, as well as apoptotic tumor cells, consistent with observed augmentation of CTL activity *in vivo* by anti-MIF, which was associated with enhanced expression of the common γ_c chain of the IL-2 receptor that mediates CD8⁺ T cell survival. CD8⁺ cells of anti-MIF treated tumor-bearing mice showed increased migration into tumors of control mice. Methods for enhancing a CTL response by inhibition of MIF are disclosed.